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Effects of Body Heating During Sleep Interruption

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Summary: This study assessed the effects that elevating body temperature had on sleep structure in the third and fourth sleep cycles, cycles typically characterized by a high propensity for REM sleep and diminished levels of delta amplitude and incidence. The sleep of eight women and two men was interrupted for 30 min on each of 3 consecutive nights following an undisturbed adaptation night. The subjects were awakened each night following the end of the second REM sleep period. On 2 nights, subjects were immersed to midthorax in water at either 34°C (TW condition) or 41°C (HW condition) for 20 min. A third interruption without immersion (NW condition) was performed to provide a second type of baseline condition. The HW condition induced a mean tympanic temperature rise of 2.5°C, that returned to baseline levels in ~60 min. Analysis of sleep patterns focused on the two sleep cycles following interruption. The mean of the two baseline conditions (TW + NW/2) was compared with the HW condition. Sleep onset latency, REM latency, REM duration, and eye movement activity in REM were unaffected by heating. Heating evoked increases in both total NREM and slow wave sleep, though these increases were delayed until the second cycle following sleep onset (i.e., appearing in the fourth, but not the third, NREM period). These were paralleled by increases in two objective measures of delta activity: integrated slow-wave amplitude (33% increase) and slow-wave density (10% increase). **Key Words:** Body temperature—Sleep—Passive heating—Interrupted sleep—Slow wave sleep—REM sleep—REM propensity.

In a previous study (1), exercise imposed during a period of sleep interruption resulted in suppression of both the phasic (eye movement activity) and tonic (duration) components of the REM period that followed (by contrast, NREM sleep was unaffected). The effects of exercise that were hypothesized to contribute to this REM suppression were (a) an increase in central noradrenergic turnover or (b) increased body temperature. While an inverse relationship between REM propensity and the normal body temperature rhythm is well established (2), it is unclear whether a reduction in REM propensity should follow an induced elevation of body temperature.

Two studies (3,4) sought to test the effects of elevating body temperature during sleep. Body temperature was elevated by pyrogens (3) in one case and by use of an electric blanket (4) in the second. While both studies reported reductions in REM sleep, a considerable sleep disturbance and reduction of NREM sleep as well suggested that the effects

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were due to discomfort induced by the manipulations and were not specific to REM sleep. In other studies, body heating preceded sleep but did not extend into it. Sauna baths were utilized, with heating occurring either in the afternoon (5) or ending an hour prior to sleep (6). In other studies, subjects experienced hot water immersion in the afternoon (7,8). Two of these studies (6,8) reported suppression of the first REM period, whereas all four reported an increase in delta sleep. The effects on the temporal distribution of delta sleep varied somewhat; i.e., in the two sauna studies the increase in delta sleep occurred primarily in the first sleep cycle whereas in the two immersion studies it was generalized across cycles.

In contrast with previous studies, we imposed heating during a period of sleep interruption just prior to the third NREM period. This design permitted heating effects to be examined during a period of sleep in which REM propensity is high (and body temperature near its circadian nadir) and delta incidence and amplitude have declined markedly from levels in the first two sleep cycles. Hence, the power to detect effects on either delta sleep or REM sleep was optimized by this design and permitted study of several unresolved issues:

(a) When heating occurs immediately prior to sleep and the individual returns to sleep with the body temperature still elevated, will any effects on REM or delta occur immediately or will effects be delayed until temperature has fallen?

(b) Since delta activity within sleep has declined considerably by the third NREM period, can heating induce an additional "surge" of slow wave sleep (SWS) and result in a net increase in delta sleep for the night? This issue bears on whether heating accelerates the processes that produce delta sleep or whether it merely changes the rate at which delta-producing mechanisms proceed in sleep.

(c) Does body heating facilitate sleep? The longer sleep onset latencies that typically follow sleep interruption provided a baseline against which any reduction in sleep latency following heating could be better detected.

METHOD

Subjects

Eight women and two men, aged 19–36 years, participated in the study. Most were graduate students or technical staff from the Institute of Environmental Stress. Medical histories and informed consent were obtained prior to their participation.

Procedures

Subjects slept in the laboratory a total of 4 nights, Monday through Thursday. Time of lights-out varied between subjects (2230–2400 h) but was consistent across nights within subjects. Subjects were awakened at the same time each morning, no later than 0800 h.

The first night was an adaptation night and no data were gathered. On each of the following 3 nights, subjects were awakened after 3 min of unequivocal stage 2 sleep (as judged by the reappearance of sleep spindles and/or K-complexes) following the second REM period. Subjects were kept awake for 30–35 min before returning to bed (second lights-out). There were three different interruption conditions. On the two immersion nights, subjects were awakened and asked to change into their swimsuits. They then proceeded to a large immersion tank (18,000 L capacity) located in a room adjacent to the sleep laboratory. A tympanic temperature probe (utilizing a Yellow Springs Thermistor #702) was inserted into the ear canal. Subjects waited ~5 min for body temperature to stabilize before climbing into the tank where they sat on a movable platform connected to a pulley. The platform's depth was adjusted until the water was just below the subject's armpits. Twenty minutes was spent in the tank during which time they conversed with the experimenter. Tympanic

temperature was recorded each minute during the immersion. On the thermoneutral water immersion night (TW) the water temperature was 34°C, a temperature selected to maintain the subject's comfort while minimizing any increase in body temperature. On the hot water immersion night (HW) the water temperature was 41°C. Pilot studies had indicated that most individuals could tolerate just 20 min of immersion at this temperature without extreme discomfort. To determine whether the immersion procedure itself affected sleep a non-immersion (NW) night was run as a second type of control condition: For the NW night the subject was awakened, room lights were turned on, and he or she conversed with the experimenter for 30 min while sitting up in bed.

The three interruption conditions were counterbalanced so that the HW condition came either second or third but never first. This resulted in four possible orderings to which subjects were randomly assigned. Testing nights were run consecutively to maintain experimental control, since our previous study (1) indicated no carryover effects of a longer (1 h) sleep interruption to the subsequent night's sleep.

Sleep recordings and scoring

Sleep recordings consisted of two electroencephalogram (EEG) channels (C3-A2, C4-A1), two electro-oculogram (EOG) channels, and one chin electromyogram (EMG) channel. Recording was performed on a Grass electroencephalograph at a chart speed of 15 mm/s with lowpass filtering at 0.3 Hz. Temperature data were collected during sleep in two subjects with a rectal thermistor (Yellow Springs Instrument Co.). Sampling was performed on a minute-by-minute basis during and following heating until temperature had returned to preheating levels.

All records were scored blind in 20-s epochs according to the usual criteria (9). Objectivity was maximized by assessing slow-wave activity in "borderline" stage 2/stage 3–4 epochs by means of an amplitude/frequency template and summation of waveforms by dial calipers.

Additionally, the raw EEG was filtered for 0.5–3 Hz delta activity by means of a specially designed lowpass filter. This was a three-stage filter with a cutoff frequency of 2.5 Hz, a 3.5 Hz half-power point, and a rolloff of 18 dB per octave. The filter output was summated by means of a Grass 7P10 integrator to provide an overall index of integrated slow-wave amplitude (ISWA) for an entire NREM period (excluding stage 1 and movement periods), as well as an index of slow-wave density (mean ISWA per 20-s epoch). These indices show a high level of intraindividual consistency across nights (D. E. Bunnell et al., unpublished observations).

Within-subject sleep comparisons were based on completed NREM and REM periods. A NREM period was considered complete if followed by at least 5 min of unequivocal REM sleep. A REM period was considered complete if followed by at least 5 min of unequivocal stage 2 sleep or SWS. Periods of REM sleep >5 min and broken by <15 min of NREM sleep were considered a single REM period. Rapid eye movements during REM periods were quantified by counting the number of 2-s periods in each 20-s epoch that contained eye movements. The total duration of EM in each REM period was determined, as well as the eye movement density (eye movement duration/REM period duration).

RESULTS

Data analysis concentrated on the two sleep cycles following sleep interruption (cycles 3 and 4), although analyses of whole-night REM and ISWA measures were performed. Comparisons of cycles 1 and 2 across nights revealed no significant differences in any REM

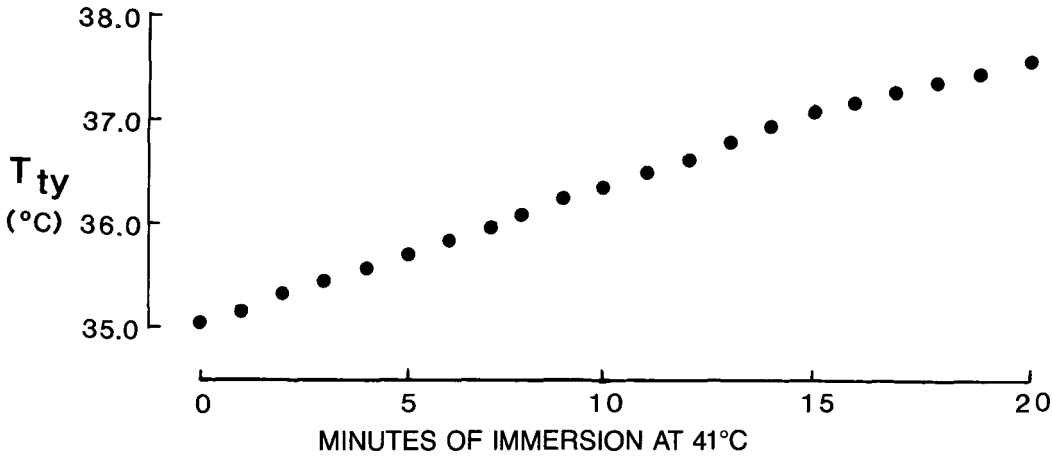

FIG. 1. Tympanic temperature changes during the 20-min of hot water immersion. Each point represents the mean of 10 subjects. T_{ty}, tympanic temperature.

or NREM variable nor any evidence of "carryover" effects. A comparison of the NW and TW baselines showed a nonsignificant tendency towards less NREM sleep following immersion. Because of the nature of the counterbalancing we utilized, comparisons of the HW condition with either baseline alone would be inappropriate. Thus, the two baselines were averaged together to provide a single baseline that was used to assess changes induced by body heating. For each sleep variable, *t* tests for matched samples were performed between baseline and the corresponding portion of the HW night. Only data from completed sleep periods, as defined previously, were used. All subjects completed the fourth NREM period, whereas only seven completed a fourth REM period in both conditions. Three subjects completed the fifth NREM period in both baseline and HW conditions, and these data were included in analyses of whole-night ISWA.

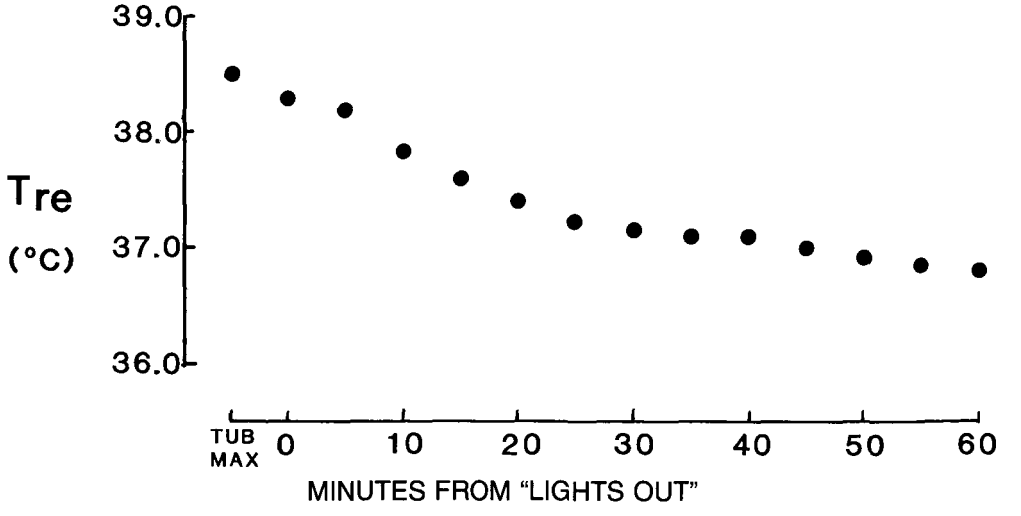

FIG. 2. Decline in rectal temperature in one subject following the 20-min hot water immersion. T_{re}, rectal temperature; Tub max, maximum temperature achieved at end of tub immersion.

TABLE 1. *Means and comparisons for nights in the third and fourth REM sleep periods*

	Condition			HW vs. (TW + NW)/2
	NW	TW	HW	
REM period 3 (n = 10)				
Latency (min)	51.5 (2.9)	44.2 (3.9)	40.7 (3.4)	NS
Duration (min)	32.9 (4.5)	28.2 (3.9)	30.0 (5.2)	NS
EM duration (s)	304 (87)	240 (61)	352 (114)	NS
EM density	0.148 (0.028)	0.132 (0.022)	0.160 (0.030)	NS
REM period 4 (n = 7)				
Duration (min)	30.0 (3.1)	41.3 (4.1)	30.5 (4.8)	NS
EM duration (s)	260 (30)	384 (71)	234 (58)	NS
EM density	0.153 (0.020)	0.164 (0.020)	0.130 (0.028)	NS
REM periods 3 and 4 (n = 7)				
Duration (min)	61.7 (5.1)	68.7 (8.2)	57.9 (6.4)	NS
Whole-night (n = 10)				
Duration (min)	90.3 (7.8)	92.0 (5.1)	87.3 (6.3)	NS

All comparisons are *t* values, test for matched samples. Standard error of the mean in parentheses. NW, nonimmersion interrupt night; TW, thermoneutral water immersion night; HW, hot water immersion night; EM, eye movements.

Temperature

Figure 1 portrays the course of tympanic temperature change during the 20 min hot water immersion. The average temperature increase was 2.5°C above the preinterruption baseline, or 1.9°C when compared with the temperature at the end of the thermoneutral immersion session. Rectal temperatures were monitored in two subjects during heating and sleep. Figure 2 portrays the course of the decline in rectal temperature of one subject after heating. Return to preinterrupt levels in this subject took ~60 min. During the same period following the thermoneutral water immersion, this subject's temperature fell from 37.0°C to 36.7°C. The differences between the end point in Fig. 1 and the initial point in Fig. 2 reflect the differences between the two measures. Although they typically parallel each other, rectal temperatures characteristically run about 1°C higher than tympanic.

Sleep latency

Body heating had no influence on sleep latency following the second lights-out. A nonsignificant tendency was observed for longer sleep latencies on the 2 immersion nights. The time from lights-out to stage 2 onset was 16.6, 22.1, and 22.1 min on NW, TW, and HW nights, respectively.

REM sleep variables

Table 1 presents the means and results of comparisons involving the third and fourth REM periods. Contrary to expectations, there were no significant changes in REM duration or eye movement activity in either of the REM periods that followed the heating session. Latency to third REM period onset was unchanged, and no differences were observed in an analysis of whole-night REM values.

NREM variables

Table 2 presents the means and results of comparisons involving NREM variables in the third and fourth sleep cycles. Means for most NREM variables tended to be lower in the TW baseline condition than in the NW baseline.

TABLE 2. *Means and comparisons for nights for NREM variables in the third and fourth sleep cycles*

	Condition			HW vs. (TW + NW)/2
	NW	TW	HW	
Cycle 3 (n = 10)				
MT (epochs)	4.2 (0.2)	2.2 (0.1)	2.9 (0.2)	NS
Stage 1 (min)	3.5 (1.2)	1.2 (0.5)	2.5 (1.8)	NS
Stage 2 (min)	40.9 (3.2)	36.2 (3.4)	36.3 (2.2)	NS
SWS (3 and 4) (min)	9.3 (3.4)	6.4 (2.6)	5.4 (2.3)	NS
Total NREM (min)	50.2 (3.1)	42.6 (3.7)	41.6 (3.0)	NS
Total ISWA (cm)	339 (45)	282 (46)	291 (37)	NS
ISWA/20 s (mm)	24.3 (2.5)	22.1 (2.3)	23.1 (1.8)	NS
Cycle 4 (n = 10)				
MT (epochs)	3.9 (0.8)	3.9 (0.5)	3.7 (0.5)	NS
Stage 1 (min)	3.7 (1.6)	1.1 (0.4)	2.6 (0.8)	NS
Stage 2 (min)	44.3 (2.8)	42.4 (2.7)	48.0 (3.6)	NS
SWS (3 and 4) (min)	7.1 (2.6)	6.7 (1.7)	12.4 (3.2)	2.46[a]
Total NREM (min)	51.4 (2.0)	49.3 (2.1)	60.4 (1.7)	4.05[b]
Total ISWA (cm)	332 (24)	320 (22)	434 (25)	5.57[c]
ISWA/20 s (mm)	22.9 (1.5)	21.5 (1.3)	24.4 (1.5)	3.86[b]
Cycles 3 and 4 (n = 10)				
Total NREM (min)	101.6 (4.1)	91.8 (5.3)	101.0 (3.1)	NS
SWS (3 and 4) (min)	16.4 (4.0)	13.6 (3.0)	16.7 (3.8)	NS
Total ISWA (cm)	671 (58)	602 (53)	732 (54)	4.72[c]
ISWA/20 s (mm)	22.0 (1.2)	21.8 (1.4)	24.1 (1.6)	4.41[b]
Whole night (n = 10)				
Total ISWA (cm)	2,270 (150)	2,217 (155)	2,376 (144)	2.86[a]

NW, nonimmersion interrupt night; TW, thermoneutral water immersion night; HW, hot water immersion night; SWS, slow wave sleep; ISWA, integrated slow-wave amplitude; MT, movement time.

[a] $p < 0.05$, two-tailed.

[b] $p < 0.01$, two-tailed.

[c] $p < 0.001$, two-tailed.

No significant changes in NREM variables appeared in the third NREM period. However, in the fourth NREM period there was significantly more SWS and a tendency toward more stage 2 sleep, resulting in a significant increase in total NREM sleep for that cycle. These increases were paralleled by significant increases in ISWA and ISWA density, 33 and 10%, respectively. Analyses performed on the combined data for cycles 3 and 4 indicated net increases in ISWA and ISWA/20 but not in SWS or NREM sleep. For the three subjects who completed a fifth NREM period, there appeared to be no further increase in delta sleep beyond those in the fourth NREM period. An analysis of whole-night ISWA values confirmed a small but significant increase (~6%) for the HW night as a whole.

Correlational analyses

Correlations were computed between ISWA changes and (a) absolute level of tympanic temperature achieved and (b) increase in tympanic temperature from beginning to end of heating. No significant relationships emerged. To assess the influence that elevated body temperature might have on REM duration, correlations were then computed between REM latency and REM duration on the HW night (reasoning that the body temperature elevations induced by heating would decline at similar rates across subjects). A tendency ($r = 0.45$, NS) was found for shorter REM latencies to be associated with shorter REM periods.

DISCUSSION

It was hypothesized that body heating might suppress REM sleep and enhance delta sleep. However, no reliable changes were observed for any REM variable following heating, indicating that disturbing the circadian temperature rhythm with an induced elevation of temperature is not sufficient to inhibit REM sleep. Indeed, there was a tendency noted for greater eye movement activity following heating. Perhaps REM would have been suppressed if body temperatures could have been sustained during sleep at the high level achieved by heating, but this would have required a different experimental approach. Nevertheless, this null finding suggests the REM suppression that followed exercise in our previous study (1) was not simply the result of increased body temperature. Although the exercise manipulation lasted longer than the heating (50 vs. 20 min), the total heat load sustained by subjects was somewhat higher in the heating session. The increase in temperature achieved by heating was >1°C higher than that during exercise, although the return to baseline temperature levels took about the same length of time in both studies. The differential effects on REM sleep induced by exercise and heating must reflect the additional physiological changes induced only by the former, i.e., increased metabolic rate and/or increases in central/peripheral noradrenergic activity. It has been shown (10) that for comparable increases in body temperature, exercise produces considerably larger increases in plasma catecholamines than does passive heating. A review of animal studies (11) indicates that heat stress involves minimal noradrenergic activation in the CNS whereas prolonged increases in norepinephrine turnover may follow exercise (12,13). If REM propensity is indeed reduced when NE turnover is increased (14), a reduction in REM would be expected following exercise but not following heating.

The increases in delta sleep are consistent with previous studies (5–8). Our study was the first to utilize objective measures of delta activity; and indeed, the changes in these indices were considerably more robust than those in manually scored SWS. [This may also reflect the problem of reduced delta amplitude in the third and fourth NREM periods where increased delta activity may be missed in manual scoring because it is likely to fall below the 75 μV criterion (9).] It is noteworthy that a 20 min heating exposure was able to produce such robust effects since the studies using hot water immersion (7,8) utilized a total of 80 min of immersion at the same water temperature. Thus, the duration of heating need not be extreme to induce a reliable increase in delta sleep.

Why does body heating produce an increase in delta sleep? Horne (15) has proposed that elevating the body temperature, either actively (as in exercise) or passively (by body heating), accelerates the waking processes that normally regulate SWS. A waking parameter proposed to regulate the requirement for delta in sleep is the cerebral metabolic rate (16). Body heating in animals lacking a carotid rete results in a corresponding increase in brain temperature as well. An increase in brain temperature should induce a proportional increase in cerebral metabolic rate (17,18) and, perhaps, a functionally accelerated form of wakefulness. Consistent with this hypothesis is the demonstration by Hoagland (19) that time is perceived as passing more rapidly when body temperature is increased by diathermy.

Jouvet (20) recently proposed that SWS is regulated by the level of serotonergic activity in waking. Animal studies are generally consistent in indicating an increase in central serotonergic activity during heat stress (21–24). These increases are associated with an increased rate of firing by serotonergic raphe neurons (24) and suggest that serotonergic pathways are involved in thermoregulation, particularly in reducing heat production and

increasing heat loss (11). If Jouvet's model is accurate, heat stress should lead to increased production of delta sleep–inducing substances.

Jouvet (20) has also made a distinction between sleep-promoting and sleep-facilitating agents. While heating clearly promotes delta sleep, it has not been determined whether it facilitates sleep as well. Our present study provides a better test of this distinction than was available from previous body heating studies, due to the much longer sleep latencies found following sleep interruption than those found at the beginning of the night. This characteristic change in sleep latencies provided greater power to detect any sleep-facilitating benefit of heating. The data show little evidence of such an influence. However, an even more powerful test could be provided by assessing the effects of body heating on individuals who typically have problems falling asleep.

Although it was not a focus of this study, some further observations were made concerning the effects of sleep interruption per se. In our previous study (1), a 1 h interruption was performed, and NREM sleep in the cycle that followed was reduced to 39 min. With one-half-hour interruption, NREM was somewhat, but not significantly, longer (50 min). We had previously speculated that this reduced NREM period following interruption was related to the time of the second sleep onset (more specifically, its position near the temperature nadir). In this study with 20 nights of baseline data available, a correlation was run against the clock time of second sleep onset. Later clock times were indeed associated with shorter REM latencies ($r = -0.61$, $p < 0.01$). Thus, the REM latency observed after sleep interruption is partially a function of the actual clock time the subject returns to bed.

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REFERENCES

1. Bunnell D, Bevier WC, Horvath SM. Sleep interruption and exercise. *Sleep* 1984;7:261–71.
2. Czeisler CA, Weitzman ED, Moore-Ede MC, Zimmerman JC, Knauer RS. Human sleep: its duration and organization depend on its circadian phase. *Science* 1980;210:1264–7.
3. Karacan I, Wolff SM, Williams RL, Hursch CJ, Webb W. The effects of fever on sleep and dream patterns. *Psychosomatics* 1968;9:331–9.
4. Karacan I, Thornby JI, Anch AM, Williams RL, Perkins HM. Effects of high ambient temperature on sleep in young men. *Aviat Space Environ Med* 1978;49:855–60.
5. Maloletnev VI, Chachanaschvilli MG. Changes in sleep structure after rapid weight reduction in steam bath. *Bull Acad Sci Georg SSR* 1979;96:690–2.
6. Putkonen PTS, Eloman E, Kotilanen PV. Increase in delta (3 + 4) sleep after heat stress in sauna. *J Clin Lab Invest* 1973;32(suppl 130):19.
7. Horne JA, Staff LHE. Exercise and sleep: body-heating effects. *Sleep* 1983;6:36–46.
8. Horne JA, Reid AJ. Night-time sleep EEG changes following body heating in a warm bath. *Electroencephalogr Clin Neurophysiol* 1984;60:154–7.
9. Rechtschaffen A, Kales A. *A manual of standardized terminology, techniques, and scoring systems for sleep stages of human subjects*. Washington DC: U.S. Government Printing Office, 1968.
10. Powers SK, Howley ET, Cox R. A differential catecholamine response during prolonged exercise and passive heating. *Med Sci Sports* 1982;14:435–9.
11. Myers R, Waller MV. Thermoregulation and serotonin. In: Essman WB, ed. *Serotonin in health and disease, vol 5*. New York: Spectrum Publishing, 1979.
12. Stone EA. Adrenergic activity in rat hypothalamus following extreme muscular exertion. *Am J Physiol* 1973;224:165–9.
13. Brown BS, Payne T, Kim C, Moore G, Krebs P, Martin W. Chronic response of rat brain norepinephrine and serotonin levels to endurance training. *J Appl Physiol* 1979;46:19–23.
14. Hartmann EL. *The functions of sleep*. New Haven: Yale University Press, 1979;106–11.
15. Horne JA. The effects of exercise upon sleep: a critical review. *Biol Psychol* 1981;12:241–90.
16. Feinberg I, Floyd TC. The regulation of human sleep: clues from its phenomenology. *Hum Neurobiol* 1982;1:185–94.
17. Hagerdab M, Harp JR, Nilsson L, Siesjo BK. The effect of induced hypothermia upon oxygen consumption in the rat brain. *J Neurochem* 1975;24:311–6.

18. Siesjo BK. *Brain energy metabolism*. Chichester: Wiley, 1978;327–32.
19. Hoagland H. Some biochemical considerations of time. In: Fraser JT, ed. *The voices of time*. New York: G. Brazillier, 1966:312–24.
20. Jouvet M. Indolamines and sleep-inducing factors. In: Borbely A, Valatx JL, eds. *Sleep mechanisms. Exp Brain Res* [suppl 8] 1984;81–94.
21. El Halawani ME, Waibel PE. Brain indole and catecholamines of turkeys during exposure to temperature stress. *Am J Physiol* 1976;230:110–5.
22. Simmonds MA. Effect of environmental temperature on the turnover of 5-hydroxytryptamine in various areas of rat brain. *J Physiol* 1970;211:93–108.
23. Corrodi H, Fuxe K, Hokfelt T. A possible role played by central monoamine neurones in thermo-regulation. *Acta Physiol Scand* 1967;71:224–32.
24. Weiss BL, Aghajanian GK. Activation of brain serotonin metabolism by heat: role of midbrain raphe neurons. *Brain Res* 1971;26:37–48.